THE SURGICAL TREATMENT OF THROMBO-EMBOLISM AND ITS SEQUELAE*

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INTRODUCTION

THE surgical treatment of thrombo-embolism must rest on the recognition of factors leading to thrombi and their propagation. The surgeon who ignores the fact that clotting of blood occurs because of slowing of venous return, intimal injury and increase in the clotting activity of the blood will simply use mechanical measures to inhibit the spreading of thrombosis. This would be very much like operating on a diabetic without the benefit of controlling his carbohydrate and fat metabolism. Surgical measures then constitute simply one phase of our therapeutic measures, which have just been so thoroughly discussed by Dr. Duryee. A combination of all such measures is highly desirable and requires an intimate teamwork of surgical and medical departments.

The literature on this fascinating subject is vast and has been reviewed annually for the last ten years by Scupham and myself in the Archives of Internal Medicine.[1] For this reason I shall limit this dis-discussion to the experience of our group. Many of the problems encountered are still unsettled and such a symposium as this helps to clarify controversial points.

FACTORS LEADING TO THROMBOSIS

Slowing of venous return. The surgeon is frequently confronted with patients whose venous return is retarded because they are put to bed, or because the operative procedure itself has diminished venous back-flow.[2] This can be conveniently measured by the ankle-to-tongue circulation time with decholin. In past years we have advocated the postoperative use of a stationary bicycle attached to the foot of the bed

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but have abandoned its use, since it is cumbersome and can not be used on a wide enough scale. Much simpler is the routine elevation of the foot of the bed on eight-inch shock blocks[3] which measurably accelerates circulation time and takes no additional nursing care. In addition it straightens out certain acute angles and compressions of the venous return, which the customary postoperative position produces. The patients are asked to move about freely in bed (barring certain obvious exceptions) and are allowed to get out of bed reasonably early. By this I mean that no extremes are used. They are neither forced to get out of bed the day after the operation, nor are they made to lie absolutely quiet for fourteen days after a herniorrhaphy. Generally speaking they should have enough sedatives for the first forty-eight hours so that they move about in bed freely and are encouraged to do deep breathing exercises, such as Dr. Eugene Pool of this city prescribed many years ago.[4] Then they are allowed to get up on the fourth or fifth day so that when the stitches are ready to be removed, they are ready to go home. With the use of non-absorbable suture material, such as silk and cotton, the hernias heal just as firmly if you let them get up the fifth as if you mobilize them the fourteenth day.

One of the most tragic types of death is that of the young man who has a herniorrhaphy and after a perfectly smooth convalescence, with no elevation of pulse and temperature, no indication of any peripheral venous thrombosis, drops dead on getting out of bed on the fourteenth day. It is difficult to escape the conclusion that a potent factor in such a patient's death is the prolonged immobilization.

Injury to the intima. The contusion, stretch or rupture of a vein obviously produces thrombosis, and these factors may later be responsible for the development of superficial varicosities. I wish to point to the not infrequent ankle edemas and tortuous perimalleolar varicosities which follow a simple sprain. Injuries to the knee-joint or to the patella, followed by rigid splinting are apt to be accompanied by a thrombosis of the popliteal vein, a result probably of injury and immobility. But injury does not have to be mechanical. Burns, frostbites notoriously produce thrombi. Certain infections, notably the pneumococcus and virus pneumonia, malaria, bacterial endocarditis, are known to be followed by thrombosis and at least part of the mechanism responsible for this is an intimal reaction, although the changes in the clotting factors are equally important. Infections, toxins may injure the intima, but by

far the most neglected phase of this problem is the allergic response of the vascular bed so clearly established for periarteritis nodosa[5] and for streptococcus infections treated with sulfanilamides.[6] For many years our group has treated Buerger's disease on the assumption that small increasing doses of typhoid vaccine desensitize the vascular bed to an unknown allergen.[7] The work on nicotine[8] and on ringworm infections[9, 10] would suggest that these are specific sensitizing factors; there are probably others.

The surgeon's interest in such vascular allergies is explained by the fact that he may have to operate in the presence of such hypersensitivity and thus precipitate a thrombosis. The situation is especially obvious in Buerger's disease, in which a minor amputation or a sympathectomy may be the starting point of widespread thromboses in which venous and lymphatic reactions may play a part. That sensitization phenomena directly affect the clotting mechanism is to be discussed presently. Certainly our best indication of such hyperactivity to date lies in changes in the tendency to thrombosis to be described presently.

Changes in the clotting mechanism. Determinations of coagulation and bleeding time are used routinely to pick out patients who suffer from hemophilia, thrombopenia, purpura and other blood dyscrasias. But with the exception of Bancroft's clotting index,[11] no attempts have been made in the past to detect "clotters" among surgical patients who might be more apt to develop postoperative, postpartum or postinfectious thromboses. Attention should be drawn to the significance of prothrombin determinations on dilute plasma,[12] to the increased heparin resistance as measured in vitro[13] and to the heparin tolerance of the patient. Since my experience is limited to this test, a brief discussion of its significance will follow.

It seems that each patient reacts individually to a certain standard dose of heparin. We chose one cubic centimeter (10 milligrams) of undiluted heparin given intravenously, and studied its effect on capillary coagulation time. The response to heparin depends on many factors some of which have been recently studied.[14] (Table I.) One measures the excess of clotting factors, thrombokinase and prothrombin by their anti-heparin action; any condition which liberates thrombokinase, such as trauma to muscle, platelet destruction, or tissue necrosis, will flatten the tolerance curve; conversely, any method which throttles the production or liberation of prothrombin such as hepatic damage, constric-

TABLE I

FACTORS INFLUENCING THE RESPONSE TO HEPARIN

Decreased sensitivity	*Increased sensitivity*
Postoperative state[1]	Sulfur compounds (sodium tetrathionate, sodium thiosulfate, sulfanilamides?)
Acute thrombosis[2]	Prostigmine (or other parasympathetic stimulants)
Buerger's disease (acute phase)	Dicoumarol[3]
Polycythemia	Digitalis (small doses)
Severe burn, sudden dehydration	Hepatic damage
Acute hemorrhage Severe trauma	
Adrenal stimulation (anxiety? fear?)	
Digitalis (toxic doses)	
Carcinomatosis	

[1] 3 to 4 days after a major operation.
[2] In any part of the vascular tree.
[3] Walker, J. and Rhoads, J. E. *Surgery*, 1944, *15*:859.

Unless otherwise stated, the observations are our own.

tion of hepatic veins (prostigmine) or which actually increases the heparin activity of the blood, such as the sulfur compounds, or raises the level of the heparin activator (albumin X of Quick[15]) will raise the tolerance curve. But most importantly there are people who perhaps at certain times or possibly consistently are hyporeactive to heparin, whereas others, a considerable number in our series (10 per cent) are hyperreactive and show symptoms of drug allergy when given heparin.

The significance of both of these groups existing in the adult surgical population is obvious. Could it be that the hyporeactors are the ones that need intensive prophylactic anticoagulant therapy, because the incidence of postoperative thrombosis is higher in this group? Such a study is now under way, but will require a rather large number of patients before it can be statistically significant. The hyperreactors are important, too, since the use of heparin in them is fraught with real danger. The bronchial spasm, the flushed face, the severe girdle or lumbar pain are mostly found in individuals who may have other

allergies. Some of these patients have never had heparin before but it must be remembered that the purest heparin contains 2 per cent nitrogen.[16]

Our present practice is to determine the heparin tolerance of every patient who is to receive heparin, since this helps in outlining the intervals between intermittent administration and also eliminates the hypersensitive individual. The prophylactic use of anticoagulants is limited to patients who have had previous thromboses, who have Buerger's disease, who have arterial or venous sutures. The treatment of thromboses by anticoagulant therapy has already been extensively discussed by Dr. Duryee.

Types of thrombosis. In his lectures on pathology Aschoff[17] distinguished between a bland thrombus due to stasis and an infectious thrombus. Great credit is due to Ochsner and DeBakey for emphasizing and popularizing these two types and coining the descriptive labels of phlebothrombosis and thrombophlebitis.[18] In a recent publication, Fowler and I[19] made the point that every thrombosis may contain elements of both of these types but in different proportions. Thus for instance a thrombosis in the calf muscles of the leg, which starts as a bland, quiet type,[20] and has embolizing quality, becomes an infectious thrombophlebitis if it ascends to the groin and encounters a latent lymphadenitis and perilymphangitis, which so often exists at the root of the limb. Then the region becomes hot, tender, a high white count and elevated temperature may appear, and the clinical picture is that of an acute iliofemoral thrombophlebitis accompanied by vasospasm since it involves the adventitial and perivenous sympathetics. It has seemed to me that with the exception of the rather rare suppurative thrombophlebitis which often produces a bacteremia and metastasizing abscesses, most thrombi are phlebitic or not depending on the existence or degree of a latent infection in the wall or the perivenous lymphatics of the vessel. Thus a thrombus produced by injecting a sclerosing solution into a varicose vein will be a bland, non-inflammatory thrombus or a markedly inflamed, red, hard cord of thrombophlebitis depending on the extent of a "resting" infection in the injected vein.[21] It seems as if this infection slumbers in the perivenous lymphatics since such a flare-up is often seen in patients who have infected ulcerations or ringworm infections in the periphery. As I have pointed out elsewhere[21] the simple, mild trauma of a venipuncture may activate

such a thrombophlebitis. It is reasonable to assume, then, that the primary source of clotting is the bland thrombus due to the process of intravascular clotting and that the infectious quality is rendered by the segmental distribution of latent infection in the perivenous lymphatics. If such a thrombophlebitis continues to pile up a static, red thrombus on top of it, may throw emboli, although the infectious head of the thrombus is well fixed to the wall of the vein.

The surgeon's interest in such distinctions lies in the fact that the inflammatory thrombophlebitis is more apt to produce a fixed thrombus but with a lot of pain, edema and vasospasm, whereas the bland, aseptic thrombus is much more silent, more insidious and more apt to break loose. However, both may merge into each other and in both the control of the clotting mechanism is of great importance.

Localization of Thrombi

There are many small, subclinical thrombi in most of us and they seem to form at segments of stagnation due to some mechanical impediment of blood flow. Neumann[22] has studied this problem extensively on cadavers. It should be noted that wherever venous return is constricted by a ligament, a tendon, a kink or a valvular stricture[23] the blood will more readily clot, although it needs some other factor such as intimal injury or increased clotting tendency to bring about the thrombus. It is likely that propagating thrombi, which become clinical when they attain a certain length, use these small phleboliths as a starting point.

The superficial saphenous system exhibits thrombi especially in varicose veins, which may remain stationary or ascend toward the sapheno-femoral junction. From the standpoint of ascending thrombosis or embolism, the thrombi of the short saphenous system are far more dangerous, because of the wide connection with the deep system, and because the varicosities of the short saphenous often represent a collateral pathway resulting from deep venous obstruction. Thrombi of superficial varicosities which represent a collateral, compensatory circulation, are very frequent. They occur in the suprapubic, or lumbar regions, in the thigh or lower leg, but the pattern of these varicosities is so characteristic that they readily point to an original deep venous obstruction. Thrombi in the plantar, supramalleolar, posterior and anterior tibial, femoral, iliofemoral, and pelvic segments give a fairly

typical clinical picture and need only be mentioned here, since a topical diagnosis is necessary to effect a surgical removal, or their elimination by division of the vein proximal to the clot.

Paravertebral Sympathetic Block

That an occlusion of any vascular segment, arterial or venous, may result in vasospasm of the collateral bed is well established. But is it really necessary to inject every case of thrombosis of the extremities paravertebrally? In iliofemoral thrombosis, the typical milk-leg, the inflammatory exudate fills the vascular sheath, directly irritates the artery to constrict, and may even completely obliterate it by spasm. One can see this during surgical explorations of such segments. At this time, and even months later when a heavy, cement-like grayish cover forms around the vein, the perivascular nerves are stimulated and diffuse painful vasoconstriction results. A paravertebral block rapidly eliminates the vasospasm and helps to decrease the edema. However, we reported several years ago that small doses of Roentgen-ray, given over the inguinal or paravertebral lymphatics, will equally be effective. Certainly the sympathetic block for the bland phlebothrombosis of the lower leg does not seem indicated, in fact it may even add to the spread of the thrombus since it relaxes not only the arterial but the venous bed.[24]

While our group has made extensive use of paravertebral blocks in arterial occlusions[25] its use in venous obstructions has always been limited. At present it is employed in the painful, edematous iliofemoral thrombosis with cold, cyanotic toes, suggesting arterial occlusion. In such cases the help obtained is spectacular. Considerable help has been obtained in chronic thrombophlebitic edemas which are painful, neuritic, vasospastic, and which are doubtless due to a continuous irritation of the vasomotor supply by the periphlebitic scarring. In acute axillary thrombosis following effort, the decrease in edema and pain is very gratifying.

Sympathetic Ganglionectomy

If paravertebral sympathetic block seems so useful, why would not a permanent interruption of the sympathetics be advisable for the chronic thrombophlebitic edemas? Such attempts have been repeatedly reported[26] but their widespread use has not followed. Attention should be called in the first place to the fact that the paravertebral lymph

glands are often enlarged, harbor infection, and that the removal of the chain therefore is not easy and may stir up infection. But more important is the finding that the indurations and ulcerations, while temporarily greatly improved during the first week of intense hyperemia which follows sympathectomy, do not seem to be greatly influenced by permanent sympathetic denervation. While sympathectomies are used extensively in our clinic for other conditions, chronic thrombophlebitic edema has not been included in our indications.

Venous Stripping

In 1937 I presented the case of two patients[27] who derived great benefit from stripping the perivenous fibrosis from the axillary and iliac veins respectively. These operations were done with the idea that most of the distress was due to a reflex vasospasm originating from the thrombosed vessel. As Leriche stated, these vessels have lost their function to conduct blood and simply act as irritable nerve plexuses maintaining reflex phenomena. It has become obvious, however, that a division of the vein with a small intervening segment is equally effective in interrupting these impulses. Perivenous stripping is equivalent to periarterial sympathectomy with its limited usefulness. It has one great advantage, however, over division in a chronic case, namely that it will not increase edema and not add to the existing difficulties of venous return.[28] The procedure of venous stripping or limited venous resection has been especially useful in handling the chronic cases of axillary thrombosis, whereas in the acute ones, a block of the dorsal sympathetics has been most effective.

Division of the Vein Proximal to Thrombosis

Division of the vein instead of simple or double ligation is always preferable since it seems to interrupt the perivenous nervous structures. However, it may not always be possible to do so because of anatomic difficulties. The first proximal divisions in the presence of thrombosis in our material were done on the saphenous system. The ligation of the saphenous vein in the presence of an acute saphenous thrombosis was advocated in 1930,[29] not only to arrest the propagation of the thrombus, but to relieve the inflamed varicosities from painful back pressure and thus permit the early mobilization of the patient with sufficient elastic support. At present, the patient who presents himself

with an acute ascending thrombosis of the saphenous vein is subjected to a typical high saphenous division. Often one finds a large saphenous bulb filled with a thrombus and occasionally one may find a loose tail of a thrombus in the iliofemoral segment. For this reason, it is advisable to aspirate the femoral vein through an eye dropper or a rectangular drinking tube[30] in case the long saphenous vein is found to be occluded at the sapheno-femoral junction. Naturally no retrograde injection should be made at this time, because even the simple division of the vein with its accompanying lymphatics may set up a marked periphlebitic reaction.

Not infrequently the acutely thrombosed saphenous varicosities are collaterals to a chronic deep venous obstruction. Even so they may be ligated, but the edema of the leg will persist and the patient will have to wear an elastic support. This does not detract from the protective value of the saphenous division against pulmonary embolism, but it must be clearly stated to the patient that a deep venous insufficiency will persist.

With the early recognition of plantar-vein[31] and deep lower leg thrombosis, the latter described in so classic a form by Homans,[32] the possibility arises of ligating the femoral vein below the profunda and thus not only exclude the thrombus from the circulation, but prevent an edema which would develop if the thrombus occluded both the superficial and deep branches of the femoral vein. When the typical symptoms of lower leg thrombosis occur in a patient who is hospitalized because of an operation, childbirth or a cardiac lesion, the ligature and division of superficial femoral vein below the profunda seems indicated. I am not convinced that this has to be done on both sides unless there are symptoms pointing to the involvement of both lower legs. In addition to Homans' dorsiflexion sign, which may not always be clear-cut, a slight filling of the dorsal veins, dependent cyanosis after a few minutes of dangling the feet and thus raising venous pressure, a slight warmth and tenseness of the calf compared with the other side are indicative of the side of involvement. Very exceptionally is phlebography used to establish the diagnosis of deep venous obstruction; while it may give a striking picture, when the lesion is clinically obvious, it fails you just exactly when you most need it. Furthermore, the injected diodrast or other opaque substance may produce thrombosis as reported by Homans[33] and also observed by

me in two unpublished cases. While we have used the visualization of blood-vessels off and on for many years, its use has always been greatly limited. At present, if the patient has thrown an embolus to the lungs and there is no indication from where it came, we visualize the deep venous circulation of the two lower extremities. This does not always give the answer; the embolus may have come from the pelvis or the right heart, or actually from the lower extremities, but the films may still be inconclusive.

While the division of the femoral vein below the profunda leaves very little residual damage, if the profunda and saphena are patent, the division of the common femoral vein, necessitated because of a thrombus in the profunda or in the iliac segments, has invariably resulted in permanent edema in our hands. In such cases the surgeon is confronted with a thrombus in the iliofemoral segment, which he can aspirate until he gets free flow of blood; then he tries to free the superficial and deep branches from thrombi as far as he can and finally divides the vein above the profunda with a patent proximal segment.[34] There are several difficulties, however, with this procedure. The proximal segment may be difficult to clear; mural thrombi may be left on the wall of the external iliac vein; the internal iliac vein may have floating thrombi in it still capable of breaking loose; the collateral circulation is not very favorable at this point.[20] For this reason Homans has suggested the ligature of the common iliac vein or even the vena cava. The latter procedure has been used some time ago on the Continent to prevent septic thrombi from entering the circulation in puerperal sepsis. Of 526 women who had a venous division for puerperal sepsis, 267 died, a mortality of 50.7 per cent, according to the collective review of Nürnberger.[35]

It has seemed to me that the age of the thrombus is of paramount importance in deciding about the level of such divisions. Obviously if the thrombus has existed at the groin for a week or more, it is organizing, it is not apt to break loose, and it is difficult to remove. The fresh, soft, floating tail of an ascending thrombus is of course the dangerous one and is mostly found when it is not expected. Our practice has gradually developed into a non-interference with the clot when it has produced a massive milk-leg, since an effective ligature would mean the level of the common iliac and since emboli from this source are not too frequent.[36] But what is most important, an adequate

anticoagulant therapy protects the patient from embolism and still does not interfere with a recanalization of the vein.

The question immediately arises: why not use anticoagulants entirely even in the case of lower-leg thrombosis, and are they as safe and effective as the interruption of the venous current. To examine this question, we have alternately used anticoagulants alone against anticoagulants with division of the vein. Division of the vein alone as used in some clinics certainly does not protect the patient from some of the sequelae of a vein ligation. Thrombi may form both in the proximal and distal segments or the previously patent collaterals of the venous system. Fowler and I have reported such sequelae in our earlier cases.[19]

If one could state that anticoagulant therapy alone would safely protect the patient from embolism one could entirely dispense with surgical interruptions. The difficulty lies, however, with the close daily control of the clotting mechanism, since inadequate control is insufficient. Of 78 patients who have received the combined heparin-dicoumarol therapy, two have shown emboli; both of them with a prothrombin level of over 70 per cent of normal, obviously not an adequate protection.

A case in point is that of Alfred O., a 35-year-old radio actor, who developed cramping of the calves followed later by dyspnea, pain in the shoulder and coughing after an exploration of his right knee for an injured semilunar cartilage. His case was not diagnosed by his attendants as a lower leg vein thrombosis followed by a pulmonary infarct. He left the hospital without any swelling but entered my service two months later with a marked, tense edema of the lower leg, which promptly stopped at the level of the knee. Homans' dorsiflexion sign was positive. The femoral vein was not tender to pressure and there was not the slightest swelling of the thigh. He was primarily admitted to see whether he could not have relief from his painful edema, which necessitated the use of cane or crutch. After elevation of the foot of the bed the swelling decreased markedly and measurements were made for an elastic stocking. However, his heparin tolerance curve was absolutely flat, meaning that 10 milligrams (1 cc.) of heparin was unable to raise his coagulation time at all. This was our warning that the clot was still active. Our routine anticoagulant therapy (heparin-dicoumarol) was immediately started but heparin could not be given, since the slight symptoms which he had from the last dose were ac-

centuated to a real sensitivity reaction with the therapeutic dose, with bronchial spasm, flushing of the face and feeling of faintness. Therefore only dicoumarol was continued at the usual doses of 300, 200, and 100 milligrams for the first three days and 100 milligrams thereafter. On the seventh day, having ingested 1000 milligrams of dicoumarol, his prothrombin level had never dropped to less than 70 per cent of normal. That night he developed the classical signs and symptoms of pulmonary embolism, for which our usual emergency measures, to be described presently, were promptly administered. Next noon his right femoral vein was tied below the profunda under sodium pentothal anesthesia, since he was known to be sensitive to novocaine. The vein was free of clots at this level and was transected. The dose of dicoumarol was doubled. The swelling of the leg, which had previously decreased on elevation, did not recur. After a painful exasperating siege with hiccoughs, which seem to follow sometimes a lower lobe infarct with diaphragmatic irritation, he left the hospital with the elastic hose, the edema well controlled.

This history seems to be a strong argument for femoral vein ligation and against anticoagulant therapy. It should be pointed out that his thrombosis was at least two months old and putting him to bed may have started up a new segment of thrombosis proximal to the old one; the value of the heparin tolerance is also apparent, since an old well-organized thrombus does not give such a flat curve. He was also obviously less responsive to dicoumarol than is usual and an alert control of his clotting mechanism may have averted the embolus. It has been my feeling for a long time that the management of the clotting mechanism should be in the hands of the internist and his staff and not the surgical staff. Finally, with the clinical picture he presented, an immediate vein ligation might have been preferable.

Here is, however, a history which presents another angle of the problem:

Miss Ellen K., a 49-year-old secretary, has been under medical care for many years because of amenorrhea, renal glycosuria, and varicosities which appeared after an appendectomy. The veins on the right showed a typical valvular defect of the saphenous system but on the left there were in addition a number of valvular defects in the communicating veins of the lower leg. In spite of multiple ligations and injections, the varicosities would recur in the left calf obviously due

to the increased pressure in the deep venous system. This patient no doubt had a chronic deep venous thrombosis in the left lower leg. During the course of five years she was seen off and on by various physicians, who diagnosed recurrent attacks of pleurisy, not suspecting their embolic origin. Finally after five such attacks a ligation of the superficial femoral vein was done; the vein was thickened but patent and so was the deep branch. The swelling of the lower leg was not influenced either way. Eight months after the ligation she developed an acute phlebitis in the persistently recurring varicosities of the calf which subsided on hot compresses and sulfanilamides. There were no more emboli but the superficial veins are painful, thickened, and their circulation has been retarded.

This history shows that femoral vein ligation, especially in the presence of well-developed collaterals, so noticeably interferes with venous return that it may predispose to later attacks of thrombosis or certainly does not prevent them. This finding, which is not the only one in our series, has impressed me with the importance of early femoral vein ligations and not in the late chronic stages as Buxton and his co-workers have recently reported.[28]

The retardation of blood flow after ligation of the femoral vein is demonstrable by determining the ankle-to-tongue circulation time with decholin. The circulation time is roughly doubled after division of the superficial and trebled after the division of the common femoral veins.

At the present writing, based on our own experience and the study of others, it is not possible to say categorically that surgical division of the vein is preferable to anticoagulant therapy. In fact, anticoagulants should always be used since when given in appropriate amounts they seem to inhibit propagation of thrombi both in the extremity or in the embolized pulmonary artery. The indication for a division of the superficial femoral vein seems clear-cut in cases of thrombosis in the lower leg. If a hospitalized patient develops cramping of the calves, pain in the sole of the foot, a Homans' sign or other symptoms of incipient deep thrombosis, the simplest and safest method is the prompt division of the superficial femoral vein. But this should be accompanied by an anticoagulant therapy, since a spreading thrombosis into the profunda or saphenous veins may result in a large, intractable edema.[19] Much less clear is the indication for the ligation of the common femoral

or common iliac veins. The former leaves a crippled venous return. The latter is a larger surgical procedure and if necessary can be combined with a lumbar sympathectomy. Recently in exposing the right common iliac artery for the aspiration of a saddle-embolus of the aorta the common iliac vein was exposed with dispatch. This is not true, however, of the left side, when a ligation of the common iliac vein can be done only by men used to the technique of vascular surgery. Actually the danger of embolism from the source is slight, once the big veins of the groin are totally occluded and begin to organize.

A special problem arises in patients who have a chronic recurrent phlebitis in the same extremity extending for many years, and leading to emboli. One patient in our series, who has had a typical milk-leg following an abdominal hysterectomy, has had four grave pulmonary infarcts within a period of eight years. In the intervals, the affected extremity remained slightly tender, warmer to the touch and swollen. Courses of sulfanilamide therapy seemed ineffective; vein ligation proximal to the thrombus was suggested several times but refused. Prolonged anticoagulant therapy with dicoumarol is impractical in her case, since the patient is difficult to control. Such a patient, in whom cumbersome superficial collaterals are no problem, certainly would be benefited by a ligation of the common iliac vein.

This is not true, however, of another group of chronic cases in whom large collaterals have developed and in whom deep venous obstruction has given way to a deep venous insufficiency, because of the destruction of the valves and a partial canalization of the old thrombus.[37] Division of the femoral vein has been advocated for such a group recently.[28] It is doubtful, however, if the elimination of this deep valvular insufficiency offsets the creation of a permanent deep venous obstruction. As it is, such collateral veins frequently thrombose later and from our experience with many hundreds of such late compensatory varices, a femoral vein ligation may definitely hamper circulation. After all, it is better to have less edema and some varicosities than to obliterate all superficial collaterals as it is proposed, which in some cases, where deep collaterals are insufficient, will increase edema.

The Excision of Thrombophlebitic Indurations

A real problem exists regarding the management of late thrombophlebitic edema. It is probable that if all cases of acute deep venous

obstruction were handled with dispatch and with an eye on preventing chronic edema, the late thrombophlebitic indurations and ulcerations would not be as prevalent. Early vein ligations, anticoagulant therapy, sympathetic block, elastic support from toes to groin by fitted stockings will do much to prevent these late sequelae. Attention should be called to the great benefit derived from the use of heparin in early edemas, since these edemas as Zimmermann and I[38] have shown both histologically and chemically, contain much protein, much fibrin, all of which is precipitated in the tissues. Such clotted plasma then is very difficult to absorb, clogs the lymphatics, sets up a connective-tissue reaction and results in the hard, brawny indurations and ulcerations so well known to you. The patches of acute lymphedema which are stirred up by cutaneous infections or foci elsewhere keep aggravating the lesion until large plaques of cement-hard tissue, with scalloped borders develop. Severe paroxysmal pain suggests the formation of neuromas in the scar. Bed rest, hot fomentations, glycerin casts, mecholyl or salt iontophoresis may be of some benefit. But the only procedure that will get rid of this chronic inflammatory fibrosis with ulceration is a thorough excision of the entire area, down to the fascia, which in our more recent experience need not be excised. The defect is then immediately covered with a split-thickness skin graft, which is secured in place both by the help of sutures and by plasma-thrombin glue which nourishes the graft and helps its early vascularization.[39] The only difficulty I have had with such grafts is late development of small thrombi under the graft, when the perforators have not been thoroughly ligated, or a break in the graft if edema of the leg is not controlled by elastic support. This method is still the best under the circumstances, but again it should be stressed that early adequate management should obviate most of these late, incapacitating sequelae.

The Surgical Treatment of Pulmonary Embolism

In a study conducted a few years ago, Jesser and I[40] found that out of 100 cases of fatal pulmonary embolism eight patients will die instantly or within 10 minutes so that no help can be given them; but 60 will live from one hour to several days after the sudden onset of symptoms. We suggested a certain routine of emergency which is posted on every floor (Table II) and which has been now employed in 45 patients with severe symptoms as recognized by the nurses. Ten

TABLE II

PULMONARY EMBOLISM

Recognition

Sudden onset of shock with rapid, weak pulse, restlessness, difficult, rapid breathing, sweating and pallor, pain in chest, fainting, collapse or unconsciousness. Apt to be in a patient who has phlebitis or is convalescing from an operation or delivery or is a known cardiac.

Emergency Treatment

By Nurse

1. Place in semi-sitting position.
2. Start oxygen by catheter or mask immediately. Tanks are on each floor.
3. Give 1/75 grain atropine sulfate, hypodermically, immediately.
4. Call intern.

By Intern

1. Give a second dose of 1/60 to 1/75 grain atropine sulfate intravenously (if previous injection of atropine has not caused flushing of face and dilation of pupil).
2. In any case give ½ grain papaverine hydrochloride intravenously.
3. Repeat atropine and papaverine three or four times a day.
4. Order portable chest film and electrocardiogram.

Note:

Morphine, adrenalin or digitalis may aggravate the condition.

Above treatment is useful even if patient is suffering from some other condition such as coronary occlusion or a cerebral vascular accident.

TABLE III

THE SURGICAL TREATMENT OF VENOUS THROMBOSES

Method of treatment	*Number of cases*	*Results*	
		Good	*Fair*
Paravertebral block[1]	78	51	27
Sympathetic ganglionectomy	2		2
Division of femoral vein[2]	25	15	10
Excision and graft[3] of thrombophlebitic induration	25	23	2
	130	89	41

[1] Done less frequently now. The large number of indifferent (fair) results are due to improper selection of cases.

[2] The ligation of common femoral vein invariably resulted in permanent edema.

[3] In 2 cases the graft broke down months or years later.

of these died and six had autopsies. All six had large totally obstructing clots in the pulmonary artery or in the right ventricle. It seems convenient to divide the treatment of pulmonary embolism into three stages. In the first stage, the reflex phenomena which operate on the heart, the bronchi, the pulmonary arterial tree and the gastrointestinal tract are interrupted by the comparatively large doses of papaverine and atropin. Oxygen is administered by a Boothby mask or a modification of the rubber mask made out of cellulose acetate, for the cyanosis or dyspnea. Some dramatic recoveries have been observed from this procedure.

If the patient survives the initial attack the second phase of treatment starts by the administration of anticoagulants. It has been shown by the postmortem studies of Belt[41] and also is clinically recognizable that the primary occluding embolus may grow proximally or distally, involving larger and larger segments of the lung so that dyspnea and the x-ray findings suggest more and more involvement of the arterial tree. These are not new emboli, but superimposed thrombi on the initially smaller embolus. Heparin-dicoumarol therapy is definitely indicated in all patients who have suffered a pulmonary embolus. It may occasionally increase the hemoptysis. It should also protect the patient from a propagating thrombus at the site of the primary blood-clot. Since approximately 40 per cent of pulmonary emboli occur without any indication of a peripheral venous thrombosis[30] a thorough search should now be made for the origin of the embolus. Unless it is in the pelvis or in the right heart, it is apt to be in the lower leg and as previously stated this can be readily excluded from the circulation by a division of the femoral vein.

In spite of the combined therapy with papaverine, atropin, oxygen, heparin and dicoumarol, some patients who made an initial recovery, slowly fail and die on the second to fourth day after the initial attack. Unquestionably they die of right heart failure, since the combined antispasmodic and anticoagulant therapy has been unable to decrease the large resistance in the pulmonary arterial bed. While the attempts to extract a pulmonary embolus in the early stages have either come too late or may have been unnecessary, such slowly fatal pulmonary emboli, as Pilcher[42] pointed out, can only be saved by pulmonary embolectomy.

The mortality statistics of such an operation are very sad. When

last studied by us, 9 out of 134 cases survived. It must be remembered, however, that these were moribund cases operated on in an emergency.

With the renewed interest in exposing the root of the large vessels which has come about with the ligation of the patent ductus arteriosus and with operating on patients who are by no means moribund, but who show a gradually increasing cor pulmonale, with its physical and electrocardiographic findings, a very limited indication for pulmonary embolectomy still exists. Pilcher has described the difficulties encountered in operating too early when the patient is on the road to recovery or too late, when the patient might have been saved by not waiting so long. Technically the operation is not too difficult. On large surgical services, the senior surgical resident should be trained on the cadaver to perform such a procedure and a team may have to be ready for 24 hours of the day waiting for an optimal time to operate.

I discussed this indication not because of any personal experience, but because of the general surgical opinion that pulmonary embolectomy is useless and should be completely discarded. Watching some of our patients die slowly in spite of conservative therapy encourages me to try such an operation in the very occasional suitable case.

Conclusions

It seems appropriate here to sum up the experience of our group with surgical measures directed against thrombo-embolism. As stated before, these are employed in conjunction with other measures, chiefly the use of anticoagulants. The indications for paravertebral block at present are the existence of a demonstrable arterial or venocapillary spasm with cyanosis, diminished pulsation, intense stocking or glove-like pain. This clot is mostly in the iliofemoral segment with perivenous inflammatory reaction, the typical iliofemoral thrombophlebitis. There seems to be no special advantage in using this block on patients suffering from the bland, quiet type of thromboses of the lower leg.

Sympathetic ganglionectomy has been done in only two patients with a chronic phlebitic induration and ulceration. While both ulcers healed, neither the edema nor the character of the thin scar bridging the defect had been influenced. The scars broke down later and had to be widely excised.

Division of the femoral vein below the profunda has been found to be an excellent measure: (1) in patients who have had a pulmonary

embolus the source of which was localized to a thrombus below the knee; (2) in patients who suddenly experienced the classical symptoms of plantar-vein or lower-leg thrombosis without any clinical involvement of the femoral segment. Such patients, as emphasized by Bauer[43] may well have a floating, non-obstructive thrombus in the femoral vein but this causes no clinical symptoms and is dangerous as far as its tendency to break loose. Division of the common femoral or common iliac vein in the presence of a typical acute milk-leg has not been done in this group except in a few cases and I am quite uncertain about its value. In the old chronic cases, with established collateral circulation and a deep venous valvular insufficiency I have not been able to see any indication for it. Ligation of the vena cava in the occasional bilateral iliac thrombosis with emboli may save life and seems to result in no additional circulatory impairment. The approach is that to a muscle-splitting extraperitoneal lumbar sympathectomy.

Excision of a chronic thrombophlebitic ulcer with induration, followed by a dermatome graft, is a very valuable procedure and saves the patient much loss of time and suffering. Naturally, the chronic edema will not be influenced and elastic support must be worn, probably continuously. (Table III)

The slowly fatal pulmonary embolus might be aspirated from the pulmonary artery surgically.

Summary

The factors responsible for the intravascular clotting of blood must be always considered and controlled even if surgical measures are indicated. These measures therefore constitute only one phase of treatment. The surgeon must try to accelerate retarded venous return, not operate in the presence of sensitization phenomena, and attempt to control the clotting mechanism by anticoagulants. The distinction between infectious thrombophlebitis and aseptic, bland phlebothrombosis is not always possible. Both may exist simultaneously in the same patient. The segmental localization of thrombi is often determined by mechanical impediments of blood flow, produced by ligaments, tendons or angulations. Thrombi are well recognizable in the saphenous, plantar, deep lower leg, iliofemoral and pelvic veins, and their treatment does depend on their location. Paravertebral sympathetic block is used in acute and chronic iliofemoral thromboses exhibiting vasospasm. Sympathetic gang-

lionectomy for venous thromboses of the extremities and their sequelae has not been especially helpful. Division of the superficial femoral vein in cases of thrombosis of the lower leg is an excellent procedure. It not only prevents more emboli from this source but inhibits the development of an ascending thrombosis with permanent edema. Divisions at a higher level have not yet been routinely adopted, since anticoagulant therapy seems at least as efficacious and probably not as productive of late edema. Excision of thrombophlebitic indurations and ulcerations, followed by a split-thickness skin graft is very useful. The surgical treatment of pulmonary embolism is most rarely indicated, but in the slowly fatal cases may save a life.

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